Amendments to the Claims:

This listing of claims will replace all prior versions, and listings of claims in the application:

Listing of Claims:

Claim 1 (original): A method for identifying a compound that modulates cell cycle arrest, the method comprising the steps of:

(i) contacting a cell comprising a target polypeptide or fragment thereof or inactive variant thereof, selected from the group consisting of flap structure specific endonuclease 1 (FEN1), protein kinase C ζ (PKC-ζ), phospholipase C-β1 (PLC-β1), protein tyrosine kinase 2 (FAK), protein tyrosine kinase 2b (FAK2), casein kinase 2 (CK2), cMET tyrosine kinase (cMET), REV1 dCMP transferase (REV1), apurinic/apyrimidinic nuclease 1 (APE1), cyclin dependent kinase 3 (CDK3), PIM1 kinase (PIM1), cell division cycle 7 kinase (CDC7L1), cyclin dependent kinase 7 (CDK7), cytokine inducible kinase (CNK), potentially prenylated protein tyrosine phosphatase (PRL-3), serine threonine kinase 2 (STK2) or (NEK4), cyclin dependent serine threonine kinase (NKIAMRE), or histone acetylase (HBO1), or fragment thereof with the compound, the target polypeptide encoded by the complement of a nucleic acid that hybridizes under stringent conditions to a nucleic acid encoding a polypeptide having an amino acid sequence selected from the group consisting of SEQ ID NO:14, 2, 4, 6, 8, 10, 12, 16, 18, 20, 22, 24, 26, 28, 30, 32, 34, and 36; and

(ii) determining the chemical or phenotypic effect of the compound upon the cell comprising the target polypeptide or fragment thereof or inactive variant thereof, thereby identifying a compound that modulates cell cycle arrest.

Claim 2 (original): The method of claim 1, wherein the chemical or phenotypic effect is determined by measuring enzymatic activity selected from the group consisting of nuclease activity, kinase activity, lipase activity, transferase activity, phosphatase activity, and acetylase activity.

Claim 3 (original): The method of claim 1, wherein the chemical or phenotypic effect is determined by measuring cellular proliferation.

Claim 4 (original): The method of claim 3, wherein the cellular proliferation is measured by assaying fluorescent marker level or DNA synthesis.

Claim 5 (original): The method of claim 4, wherein DNA synthesis is measured by ^{3}H thymidine incorporation, BrdU incorporation, or Hoescht staining.

Claim 6 (original): The method of claim 4, wherein the fluorescent marker is selected from the group consisting of a cell tracker dye or green fluorescent protein.

Claim 7 (original): The method of claim 1, wherein modulation is activation of cell cycle arrest.

Claim 8 (original): The method of claim 1, wherein modulation is activation of cancer cell cycle arrest.

Claim 9 (original): The method of claim 1, wherein the host cell is a cancer cell.

Claim 10 (original): The method of claim 9, wherein the cancer cell is a breast, prostate, colon, or lung cancer cell.

Claim 11 (original): The method of claim 9, wherein the cancer cell is a transformed cell line.

Claim 12 (original): The method of claim 11, wherein the transformed cell line is A549, PC3, H1299, MDA-MB-231, MCF7, or HeLa.

Claim 13 (original): The method of claim 9, wherein the cancer cell is p53 null or mutant.

Claim 14 (original): The method of claim 9, wherein the cancer cell is p53 wild-type.

Claim 15 (original): The method of claim 1, wherein the polypeptide is recombinant.

Claim 16 (original): The method of claim 1, wherein the polypeptide is encoded by a nucleic acid comprising a sequence of SEQ ID NO:13, 1, 3, 5, 7, 9, 11, 15, 17, 19, 21, 23, 25, 27, 29, 31, 33, or 35.

Claim 17 (original): The method of claim 1, wherein the compound is an antibody.

Claim 18 (original): The method of claim 1, wherein the compound is a small organic molecule.

Claim 19 (original): The method of claim 1, wherein the compound is an antisense molecule.

Claim 20 (original): The method of claim 1, wherein the compound is a peptide.

Claim 21 (original): The method of claim 20, wherein the peptide is circular.

Claim 22 (original): The method of claim 1, wherein the compound is an siRNA molecule.

Claim 23 (original): A method for identifying a compound that modulates cell cycle arrest, the method comprising the steps of:

(i) contacting a cell comprising a target polypeptide or fragment thereof or inactive variant thereof, selected from the group consisting of flap structure specific endonuclease 1 (FEN1), protein kinase C ζ (PKC-ζ), phospholipase C-β1 (PLC-β1), protein tyrosine kinase 2 (FAK), protein tyrosine kinase 2b (FAK2), casein kinase 2 (CK2), cMET tyrosine kinase (cMET), REV1 dCMP transferase (REV1), apurinic/apyrimidinic nuclease 1 (APE1), cyclin dependent kinase 3 (CDK3), PIM1 kinase (PIM1), cell division cycle 7 kinase (CDC7L1), cyclin dependent kinase 7 (CDK7), cytokine inducible kinase (CNK), potentially prenylated protein tyrosine phosphatase (PRL-3), serine threonine kinase 2 (STK2) or (NEK4), cyclin dependent serine threonine kinase (NKIAMRE), or histone acetylase (HBO1), or fragment thereof with the compound, the target polypeptide encoded by the complement of a nucleic acid that hybridizes under stringent conditions to a nucleic acid encoding a polypeptide having an amino acid sequence selected from the group consisting of SEQ ID NO:14, 2, 4, 6, 8, 10, 12, 16, 18, 20, 22, 24, 26, 28, 30, 32, 34, and 36; and

(ii) determining the physical effect of the compound upon the target polypeptide or fragment thereof or inactive variant thereof; and

(iii) determining the chemical or phenotypic effect of the compound upon a cell comprising the target polypeptide or or fragment thereof or inactive variant thereof, thereby identifying a compound that modulates cell cycle arrest.

Claim 24 (original): A method of modulating cell cycle arrest in a subject, the method comprising the step of administering to the subject a therapeutically effective amount of a compound identified using the method of claim 1.

Claim 25 (original): The method of claim 24, wherein the subject is a human.

Claim 26 (original): The method of claim 25, wherein the subject has cancer.

Claim 27 (original): The method of claim 24, wherein the compound is a small organic molecule.

Claim 28 (original): The method of claim 24, wherein the compound is an antisense molecule.

Claim 29 (original): The method of claim 24, wherein the compound is an antibody.

Claim 30 (original): The method of claim 24, wherein the compound is a peptide.

Claim 31 (original): The method of claim 30, wherein the peptide is circular.

Claim 32 (original): The method of claim 24, wherein the compound is an siRNA molecule.

Claim 33 (original): The method of claim 24, wherein the compound inhibits cancer cell proliferation.

Claim 34 (original): A method of modulating cell cycle arrests in a subject, the method comprising the step of administering to the subject a therapeutically effective amount of a target polypeptide or fragment thereof or inactive variant thereof, selected from the group consisting of flap structure specific endonuclease 1 (FEN1), protein kinase C ζ (PKC-ζ), phospholipase C-β1 (PLC-β1), protein tyrosine kinase 2 (FAK), protein tyrosine kinase 2b (FAK2), casein kinase 2 (CK2), cMET tyrosine kinase (cMET), REV1 dCMP transferase (REV1), apurinic/apyrimidinic nuclease 1 (APE1), cyclin dependent kinase 3 (CDK3), PIM1 kinase (PIM1), cell division cycle 7 kinase (CDC7L1), cyclin dependent kinase 7 (CDK7), cytokine inducible kinase (CNK), potentially prenylated protein tyrosine phosphatase (PRL-3), serine threonine kinase 2 (STK2) or (NEK4), cyclin dependent serine threonine kinase

(NKIAMRE), or histone acetylase (HBO1), or fragment thereof with the compound, the target polypeptide encoded by the complement of a nucleic acid that hybridizes under stringent conditions to a nucleic acid encoding a polypeptide having an amino acid sequence selected from the group consisting of SEQ ID NO:14, 2, 4, 6, 8, 10, 12, 16, 18, 20, 22, 24, 26, 28, 30, 32, 34, and 36.

Claim 35 (original): A method of modulating cell cycle arrest in a subject, the method comprising the step of administering to the subject a therapeutically effective amount of a nucleic acid encoding a target polypeptide or fragment thereof or inactive variant thereof, selected from the group consisting of flap structure specific endonuclease 1 (FEN1), protein kinase C ζ (PKC-ζ), phospholipase C-β1 (PLC-β1), protein tyrosine kinase 2 (FAK), protein tyrosine kinase 2b (FAK2), casein kinase 2 (CK2), cMET tyrosine kinase (cMET), REV1 dCMP transferase (REV1), apurinic/apyrimidinic nuclease 1 (APE1), cyclin dependent kinase 3 (CDK3), PIM1 kinase (PIM1), cell division cycle 7 kinase (CDC7L1), cyclin dependent kinase 7 (CDK7), cytokine inducible kinase (CNK), potentially prenylated protein tyrosine phosphatase (PRL-3), serine threonine kinase 2 (STK2) or (NEK4), cyclin dependent serine threonine kinase (NKIAMRE), or histone acetylase (HBO1), or fragment thereof with the compound, the target polypeptide encoded by the complement of a nucleic acid that hybridizes under stringent conditions to a nucleic acid encoding a polypeptide having an amino acid sequence selected from the group consisting of SEQ ID NO:14, 2, 4, 6, 8, 10, 12, 16, 18, 20, 22, 24, 26, 28, 30, 32, 34, and 36.

Claim 36 (currently amended): A CK2-specific siRNA molecule comprising the sequence AACATTGAATTAGATCCACGT (SEQ ID NO:37), wherein the siRNA molecule is from 21 to 30 nucleotide base pairs in length.

Claim 37 (currently amended): The CK2-specific siRNA molecule of claim 36 consisting of the sequence AACATTGAATTAGATCCACGT (SEQ ID NO:37) and its complement as active portion.

Claim 38 (currently amended): A method of inhibiting expression of a CK2 gene in a cell, the method comprising contacting the cell with a CK2-specific siRNA molecule comprising the sequence AACATTGAATTAGATCCACGT (SEQ ID NO:37), wherein the siRNA molecule is from 21 to 30 nucleotide base pairs in length.

Claim 39 (currently amended): A PIM1-specific siRNA molecule comprising the sequence AAAACTCCGAGTGAACTGGTC (SEQ ID NO:38), wherein the siRNA molecule is from 21 to 30 nucleotide base pairs in length.

Claim 40 (currently amended): The PIM1-specific siRNA molecule of claim 39 consisting of the sequence AAAACTCCGAGTGAACTGGTC (SEQ ID NO:38) and its complement as active portion.

Claim 41 (currently amended): A method of inhibiting expression of a PIM1 gene in a cell, the method comprising contacting the cell with a PIM1-specific siRNA molecule comprising the sequence AAAACTCCGAGTGAACTGGTC (SEQ ID NO:38), wherein the siRNA molecule is from 21 to 30 nucleotide base pairs in length.

Claim 42 (currently amended): An ~~Hbo1-specific~~ HBO1-specific siRNA molecule comprising the sequence AACTGAGCAAGTGGTTGATTT (SEQ ID NO:39), wherein the siRNA molecule is from 21 to 30 nucleotide base pairs in length.

Claim 43 (currently amended): The ~~Hbo1-specific~~ HBO1-specific siRNA molecule of claim 42 consisting of the sequence AACTGAGCAAGTGGTTGATTT (SEQ ID NO:39) and its complement as active portion.

Claim 44 (currently amended): A method of inhibiting expression of an ~~Hbo1~~ HBO1 gene in a cell, the method comprising contacting the cell with an ~~Hbo1-specific~~ HBO1-specific siRNA molecule comprising the sequence AACTGAGCAAGTGGTTGATTT (SEQ ID NO:39), wherein the siRNA molecule is from 21 to 30 nucleotide base pairs in length.